Late myocardial salvage: time to recognize its reality in the reperfusion therapy of acute myocardial infarction

Albert Schömig¹,²*, Gjin Ndrepepa¹,², and Adnan Kastrati¹,²

¹Deutsches Herzzentrum, Technische Universität, Lazarettstr. 36, Munich, Germany; and ²1. Medizinische Klinik rechts der Isar, Technische Universität, Munich, Germany

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The prevailing opinion in the reperfusion therapy of patients with acute myocardial infarction (AMI) is that the benefit of reperfusion is mostly confined to the first 12 h after the symptom onset. This opinion is based on the results of the prior megatrials of thrombolytic therapy and the experimental studies. Thrombolytic studies have unequivocally proven that the efficacy of thrombolysis to salvage ischaemic myocardium is drastically reduced with the increase in the time-to-treatment interval. A relatively large number of patients present beyond the limit efficacy of thrombolysis and are considered ineligible for this reperfusion modality. Recent experimental and clinical evidence indicates that a large amount of viable myocardium is still present in the area at risk in patients with AMI presenting late after symptom onset and considered ineligible for thrombolysis. In this review, we summarized the existing data demonstrating that this viable myocardium is salvageable given the primary percutaneous coronary intervention (PCI) is used as a reperfusion therapy. By emphasizing this fact, we do not mean to contest the concept of time dependence of myocardial necrosis following coronary occlusion and time dependence of efficacy of interventions performed early (within 2–3 h) after symptom onset or to dissuade the early coronary interventions in patients with AMI. Instead, we strongly recommend the primary PCI in patients with AMI presenting late after onset of myocardial ischaemia.

Magnitude of the problem

The current paradigm in the reperfusion therapy of acute myocardial infarction (AMI) is that the benefit of reperfusion in patients with ST-segment elevation AMI is confined to the first 12 h after the symptom onset. The benefit of reperfusion therapy beyond 12 h from the onset of symptoms is considered to be minimal and largely not attributable to myocardial salvage. This concept has been echoed by the current guidelines that favour acute reperfusion therapy in patients with AMI beyond 12 h from symptom onset and considered ineligible for thrombolysis. In this review, we summarized the existing data demonstrating that this viable myocardium is salvageable given the primary percutaneous coronary intervention (PCI) is used as a reperfusion therapy. By emphasizing this fact, we do not mean to contest the concept of time dependency of myocardial necrosis following coronary occlusion and time dependence of efficacy of interventions performed early (within 2–3 h) after symptom onset or to dissuade the early coronary interventions in patients with AMI. Instead, we strongly recommend the primary PCI in patients with AMI presenting late after onset of myocardial ischaemia.

Roots of the current paradigm of the reperfusion therapy

The clinical recognition that coronary artery thrombosis leads to irreversible myocardial damage that develops in a time-dependent fashion provided the basis for the use of reperfusion therapy in patients with AMI. The data obtained from experimental studies and megatrials of thrombolytic therapy gave rise to the concept of time dependency of
the benefit of the reperfusion therapy in AMI. Experimental studies in dogs by Reimer et al.10 have demonstrated that coronary occlusion results in myocardial necrosis that progresses gradually and that, in general, is complete about 6 h after the onset of occlusion. The authors emphasized the fact that cell death after coronary occlusion occurs early in the subendocardial zone and that about half of the ischaemic myocardium that is necrotic at 24 h has already died by 40 min of coronary occlusion. The cell death occurs more slowly in the mid- and subepicardial myocardium and about one-third of ischaemic myocardium is salvageable at 3 h. By 6 h, the amount of salvageable myocardium is minimal.10 The GISSI (Gruppo Italiano per lo Studio della Streptochinasi nell’ Infarto Miocardico) trial showed that patients with AMI who were treated with streptokinase within the first hour of the symptom onset had a 51% reduction in mortality. The benefit in mortality reduction reduced to 26% at 3 h and to 20% if streptokinase was applied between 3 and 6 h from the onset of symptoms.11 In the same line, ISIS-2 (Second International Study of Infarct Survival) trial demonstrated a 37% reduction in mortality by thrombolytic therapy applied up to 3 h, a 24% reduction between 3 and 6 h, and a 17% reduction in mortality if thrombolysis was applied beyond 6 h from the onset of symptoms.12 The time dependency of the efficacy of thrombolysis was further reinforced by the Fibrinolytic Therapy Trialist (FTT) collaborative group13 and the Global Utilization of Streptokinase and Tissue Plasminogen for Occluded Coronary Arteries (GUSTO-1) trial14 which unequivocally confirmed that the greatest reduction in mortality in patients with AMI was achieved if thrombolytic therapy was administered within the first hours from the onset of symptoms. These studies11-13 and two others, the EMERAS (Estudio Multi-centrico Estreptoquinasa Republicas de America del Sur) trial15 and the LATE (Late Assessment of Thrombolytic Efficacy) trial16 showed a mortality benefit from thrombolytic therapy up to 12 h from the onset of symptoms in patients with AMI. The EMERAS and LATE trials did not show a benefit if thrombolytic therapy was applied between 12 and 24 h after the symptom onset in patients with AMI. The results of these studies laid down the foundations of the current paradigm in the reperfusion therapy in patients with AMI.

Mechanistically, key to the understanding of the benefits in mortality reduction has been the demonstration of a reduction in the infarct size, estimated by enzyme measurements17 or 201Tl scan18 and a preservation of the left ventricular function17 in patients receiving thrombolysis early after symptom onset. The restoration of the blood flow in the infarct-related artery is the main mechanism underlying the benefits of thrombolysis. The GUSTO-1 angiographic substudy showed a strong correlation between the 90 min patency of the infarct-related artery with the reduction in the 30-day mortality after AMI.19 Recent scintigraphic studies with 99mTc-sestamibi single-photon emission computed tomography provided further insight into the mechanism of time-dependent efficacy of thrombolysis. They showed a progressive decrease in the amount the myocardium salvaged by thrombolysis with longer time-to-treatment intervals.20,21

Differences between experimental and clinical AMI

It has been estimated that myocardial viability which implies preservation of the basic cellular functions and mitochondrial and membrane integrity is maintained at about 20% of total coronary flow.22 Thus, assuming a normal resting blood flow rate of 1 mL/min/g of tissue, flow rates ≥0.2 mL/min/g of myocardial tissue should be compatible with myocardial cell survival.22 Mechanistically, at least 20% of normal coronary blood flow is required to effectively wash out the lactate and protons from the vicinity of ischaemic myocardial cells so that anaerobic glycolysis, the main and vital source of energy in the setting of myocardial ischaemia, can continue. Although, in essence, severe myocardial ischaemia leading to myocardial necrosis after coronary artery occlusion, experimental or spontaneously occurring in humans, has many common aspects, there are also substantial differences between them.

Experimental studies have shown that total coronary occlusion is associated with a drastic reduction of coronary blood flow resulting in severe myocardial ischaemia.10,23,24 Typically, experimental AMI is produced by fixed ligation or total occlusion of the coronary arteries. Even in the setting of total and fixed coronary occlusion which implies no antegrade blood flow in the occluded artery and no possibility of spontaneous re-opening, some residual blood flow is always present in the ischaemic area following experimental coronary occlusion.23,24 Moreover, experimental studies have shown that there is a gradual recovery of blood flow to the ischaemic region with a two-fold increase at 96 h compared with coronary blood flow immediately after occlusion.23 Considerable heterogeneity in the distribution of the residual blood flow following coronary artery occlusion has been shown experimentally.24 In general, there is a gradient of coronary flow reduction with central zones and subendocardial zones being most deprived from the coronary blood flow, whereas the outer and subepicardial areas still retaining a residual flow compatible with viability. It has been assumed that coronary flow rates as low as 0.05–0.1 mL/min/g of tissue in the central core region of the ischaemia may be sufficient to maintain the viability in the non-contracting tissue.23 Thus, low-flow ischaemia rather than no-flow ischaemia characterizes the extensive areas of ischaemic myocardium following coronary artery occlusion.

Several factors may increase the residual blood flow in the ischaemic area following spontaneous coronary occlusion or the amount and speed of necrosis progression in human AMI compared with animal models of AMI. First, in a sizable portion of patients with AMI, coronary occlusion may be not complete. Not rarely, the human AMI has a stuttering course with intermittent occlusion and re-canalization.25 Previous angiographic studies of patients with AMI have indicated that, at the time of angiography, infarct-related artery was not totally occluded in up to one-third of patients.9,25 In 33.8% patients with AMI presenting within 12 h from the symptom onset, residual antegrade blood flow in the infarct-related artery was found.26 The proportion of patients with residual blood flow in the infarct-related artery increased to 50.5% in patients with AMI presenting between 12 and 48 h from symptom onset.27 Preservation of residual blood flow in the infarct-related artery was found to be associated with a reduction in the infarct size,28,29 better left ventricular function, and more favourable clinical outcome30 compared with total occlusion. The preservation of antegrade blood flow has been demonstrated to be associated with the
preservation of fatty acid metabolism in the area at risk in patients with anterior AMI.31 It has been estimated that the beneficial effects of spontaneous anterograde blood flow in the infarct-related artery are superior to those achieved by early reperfusion.31,32 Secondly, coronary collateral circulation may be an important factor that preserves coronary blood flow and maintains viable myocardium after coronary occlusions and is a primary determinant of the left ventricular functional recovery following late mechanical reperfusion.31,34 Previous studies have indicated that chronic myocardial ischaemia due to critical coronary narrowing stimulates the development of collateral circulation to the ischaemic region.35,36 Upon coronary artery occlusion, preformed collateral vessels may supply considerable amount of blood sufficient to avoid acute necrosis. Thus, the residual anterograde blood flow in the infarct-related artery and/or collateral circulation may secure a residual blood flow that may be substantially greater in human AMI compared with experimental AMI produced by fixed coronary occlusion in animal models. Thirdly, repetitive myocardial ischaemia results in preconditioning37 which increases the resistance of myocardium to ischaemia, reduces the speed with which ischaemic myocardium succumbs to necrosis, and thus prolongs the time during which myocardium remains viable after coronary occlusion.

These factors may maintain a residual blood flow compatible with cell survival over extensive parts of the area at risk and thus may explain the presence of viable myocardium well beyond the time window of viability deduced by experimental studies of AMI. There is considerable clinical evidence on the existence of viable myocardium hours to days after coronary occlusion. Studies with positron emission tomography performed within 3 days of the symptom onset in patients with anterior AMI have shown that viability was maintained in half of the acutely infarcted segments.38 Persistence of viable myocardium has been demonstrated by experimental39 and clinical studies34,40–42 and related to preservation of the residual blood flow to the area at risk. Other studies have demonstrated the existence of viable but stunned or hibernating myocardium within the area at risk43,44 and that time window to salvage viable myocardium and ameliorate left ventricular function may be extended to weeks, given a collateral circulation is present.45 Imaging studies with fluorodeoxiglucose46 or rest-distribution thallium47 have demonstrated that residual viable myocardium following AMI may act as an unstable substrate for further adverse events including increased mortality unless revascularized. Thus, all these studies indicate that viable myocardium is present at the site of infarction even days to weeks after symptom onset and this viable tissue may be salvaged given that an effective reperfusion strategy is used. If patients do not undergo reperfusion, the viable myocardium dies gradually through apoptosis, undergoes hibernation, and provides an unstable substrate that predisposes for adverse cardiovascular events.

**Why thrombolysis cannot salvage myocardium when used late after symptom onset?**

Thrombolysis is frequently used as reperfusion therapy in patients with AMI. When applied within the first 12 h from the symptom onset, thrombolysis results in myocardial salvage and mortality reduction in patients with ST-segment elevation AMI.11–13 There are numerous factors, however, that may explain the failure of thrombolysis to salvage ischaemic myocardium if applied late after symptom onset. Even if timely applied, thrombolysis has limited capability to restore optimal blood flow in the infarct-related artery. The GUSTO-1 angiographic substudy showed that blood flow in the infarct-related artery is restored in only 85% of patients receiving thrombolytic therapy and that only half of them regain a normal coronary blood flow.19 A suboptimal blood flow in the infarct-related artery is associated with reduced myocardial salvage and worse short-term and long-term survival.46 One of the greatest disadvantages of thrombolytic therapy is its time dependence of efficacy, meaning that the efficacy of thrombolysis declines rapidly with the increase in the time-to-treatment interval. The Thrombolysis in Myocardial Infarction (TIMI) investigators have reported a decrease in TIMI flow grade 3 from 45% if streptokinase was applied at 2–4 h to 17% if streptokinase was applied >6 h after the symptom onset.49 The time dependency in the restoration of coronary blood flow in the infarct-related artery by thrombolysis seems to be reflected in the mortality benefit as well. In the GUSTO-1 trial, the 30-day mortality increased from 5.5% if treatment was initiated ≤2 h to 9% if treatment was initiated >4 h from symptom onset.50 One factor that may explain the failure of thrombolysis to open occluded coronary arteries may relate to maturation of thrombi which become more resistant to thrombolytic agents. Experimental studies have demonstrated that older thrombi are more resistant to thrombolysis due to continuation of fibrin polymerization.51 Moreover, the increased availability of plasminogen activator inhibitor type-1 (PAI-1) released from platelets with time may also reduce efficacy of fibrinolytic agents and lead to resistance to thrombolysis.52 Other factors also may contribute to failure of thrombolysis to salvage myocardium. Thrombolysis is not expected to influence positively collateral circulation which is another major determinant of the myocardial viability and the late myocardial salvage. The increased frequency of myocardial rupture with increasing time-to-treatment interval with thrombolytic therapy has been described.53 Finally, cyclic variations and instability in the coronary blood flow in the infarct-related artery with frequent reclosures54 as well as possible platelet activation by thrombolysis55,56 are factors that may reduce the efficacy of thrombolysis in general. These data show that thrombolysis has a limited power, if at all, to salvage myocardium in patients with AMI presenting late after symptom onset. This may be the reason why the current guidelines discourage thrombolytic reperfusion therapy in this setting.1,2

**Advantages of mechanical reperfusion in salvaging myocardium in late-coming patients with AMI**

In patients with AMI, primary percutaneous coronary intervention (PCI) has advantages over thrombolysis in reducing short-term and long-term major adverse clinical events including mortality.57 Available evidence suggests that
primary PCI is superior to thrombolysis in all studied time intervals. A recent report from the Primary Coronary Angioplasty vs. Thrombolysis (PCAT)-2 Trials’ Collaborative Group has demonstrated that primary PCI is superior to pharmacological thrombolysis in terms of reduction of the 30-day incidence of major adverse cardiac events and that the absolute reduction in mortality by PCI widened over time from 1.3% within the first hour to 4.2% in >6 h after symptom onset.58

The first line of evidence in favour of mechanical reperfusion comes from recent clinical studies of patients with AMI treated by primary angioplasty reporting an independence of clinical outcome from the time-to-treatment interval.59–62 In a recent analysis including 2635 patients enrolled in 10 randomized trials of primary angioplasty vs. thrombolytic therapy, Zijlstra et al.59 concluded that with increasing time-to-presentation interval, major adverse cardiac event rates increased after thrombolysis but remained relatively stable after angioplasty. Data from the National Registry of Myocardial Infarction (NRMI) showed that there is no association between symptom onset-to-balloon time and survival in a cohort of 27 080 consecutive patients with AMI who were treated with primary angioplasty.60 The authors of this study hypothesized that the outcome in patients presenting to the hospital late after symptom onset (after 6 to 12 h) reflects a survivor–cohort effect. Other studies have demonstrated that primary PCI within first 2 h is associated with lower mortality compared with primary PCI performed after 2 h of presentation; beyond 2 h, both short- and long-term mortality in patients undergoing PCI is independent from time-to-reperfusion interval.61,62 Another important source of evidence in favour of mechanical reperfusion comes from scintigraphic studies conducted to test efficacy of coronary reperfusion.20,21,27,41,42 In an analysis of the Stent versus Thrombolysis for Occluded coronary arteries in Patients with Acute Myocardial Infarction (STOPAMI) trials that included patients up to 12 h from the symptom onset, we demonstrated that longer symptom onset-to-treatment interval is associated with worsened myocardial salvage among patients treated with thrombolysis but not among those treated with coronary stenting.61 Myocardial salvage index, an estimate of amount of myocardium salvaged by reperfusion therapy was reduced markedly with longer time-to-treatment intervals only in patients treated with thrombolysis but it remained high and relatively constant in those treated with coronary stenting (Figure 1). The Beyond 12 hours Reperfusion Alternative Evaluation (BRAVE-2) trial showed that mechanical reperfusion significantly reduced the infarct size in patients with AMI presenting 12–48 h from the symptom onset, a finding that could be explained by the ability of mechanical reperfusion to salvage myocardium even in this group of latecomers after AMI onset.61 In patients presenting beyond 12 h from the symptom onset, 44% of the initial area at risk was still salvageable by primary PCI.63 Even when used as a revascularization means in patients with AMI after failed thrombolysis, mechanical reperfusion salvaged from 25 to 35% of initial myocardial area at risk64 which may provide mechanistic basis of favourable clinical outcome of mechanical reperfusion after failed thrombolysis.65 Time dependency of myocardial salvage by thrombolysis and primary PCI is shown in Figure 2.

Previous studies including the Thrombolysis and Angioplasty in Myocardial Infarction-6 (TAMI-6) trial,66 the Total Occlusion Post-Myocardial Infarction Intervention Study (TOMIIS) pilot study,67 the Open Artery Trial (TOAT) study,68 the Desobstruction Coronnaire en Post-Infarctus (DECOPI) trial,69 and a study by Horie et al.70 have performed angioplasty up to several weeks after AMI onset. The rationale of these studies was based on the open artery hypothesis, according to which most of the benefits of recanalization are independent from myocardial salvage. In the same line, the ongoing Occluded Artery Trial (OAT) is investigating whether opening an occluded coronary artery 3–28 days after AMI will improve clinical outcome.71 Notably different, the BRAVE-2 trial addressed the issue of the benefit of primary PCI in patients with AMI presenting between 12 and 48 h from symptom onset in terms of myocardial salvage.

Although, it has not been investigated, the issue of an emergency intervention in latecomers with AMI is intuitively not as essential as for patients with AMI presenting early after symptom onset. This issue, however, deserves further investigation in specifically designed studies. A strategy of facilitated PCI, a combination of thrombolysis and PCI, has been recommended to bridge the delay between presentation of patients and mechanical opening of the infarct-related arteries.72 Recent data, however, have not provided support for the use of this strategy in patients with AMI presenting within 12 h from the symptom onset.73,74 Even less is expected from the use of this strategy in patients with AMI presenting after 12 h from the symptom onset.
onset due to the known lack of efficacy of thrombolysis beyond this time limit.13,15,16

Interestingly, in patients with AMI who were treated with primary balloon angioplasty, the NRMI data indicated the existence of an association between the door-to-balloon time and the survival.60 Several factors, however, clustered in the subgroups of patients with the longer door-to-balloon time may explain the association of the worse clinical outcome with longer-door-to-balloon time. Apart being a part of the symptom onset-to-balloon time, the door-to-balloon time is an indicator of patients’ characteristics75 and experience of institution providing the primary PCI.76 Data from the NRMI showed that longer door-to-balloon times were encountered in patients with older age, female sex, non-white race, and complex medical histories. Door-to-balloon delay depended heavily on the hospital characteristics as well. Thus, transfer for primary PCI, presentation at night and treatment at lower volume facilities were evidenced as strong independent predictors of longer-door-to-balloon interval.75 A recent report from the National Registry of Myocardial Infarction convincingly showed that greater experience with primary PCI is associated with lower in-hospital mortality and shorter door-to-balloon times in patients with ST-segment elevation AMI treated with primary PCI.76

Clear evidence is available that mechanical reperfusion restores optimal epicardial blood flow (TIMI flow grade 3) in majority of patients with AMI. This has been evidenced in a study by Brodie et al.62 who reported restoration of TIMI flow grade 3 in 93% of patients in a series of 1352 patients with AMI, independent of ischaemia duration. During mechanical reperfusion, balloon angioplasty, and/or stenting, considerable mechanical force is used to dissolve the culprit coronary thrombi and dilate stenotic coronary arteries. Thus, age of thrombus per se seems not to exert any important influence on the capability of mechanical reperfusion to restore epicardial blood flow in patients with AMI. Likewise, balloon angioplasty and/or stenting restores blood flow in coronary arteries occluded by plaque expansion rather than thrombus formation, a state known to be associated with failed thrombolysis. Mechanical reperfusion with adjunct use of current peri-procedural antithrombotic therapy results in durable restoration of coronary flow in the infarct-related artery showing substantially less propensity for cyclic oscillations in the restored blood flow or re-closures. If coronary stenting is used, it seals the intimal dissections that occur by balloon angioplasty which further increases the stability of restored coronary flow.

Finally, characteristics of patients with AMI coming late after symptom onset may provide some clues for explaining the benefit of mechanical reperfusion. Previous studies have demonstrated that patients with AMI presenting late after symptom onset are older, have more often co-morbidities, and have a more adverse cardiovascular risk profile than patients presenting earlier after symptom onset.77 The associated co-morbidities and the worse cardiovascular risk profile may be one factor that may mask the benefits of mechanical reperfusion due to myocardial salvage, and the unfavourable outcome after coronary intervention may erroneously be attributed solely to the longer time-to-reperfusion interval. The BRAVE-2 trial showed that primary PCI is safe in patients with AMI presenting between 12 and 48 h after the symptom onset and the procedure-related complications were well in the range of complications rate for PCI procedure in AMI patients in general. This finding is also supported by a large series of patients with AMI treated with PCI >12 h from symptom onset and included in the National Registry of Myocardial Infarction 2.78 Nevertheless, future larger clinical trials will better define the risk–benefit ratio of this treatment strategy. The group of latecomers may include patients

Figure 2  Time dependency of myocardial salvage expressed as percentage of initial area at risk. The initial parts of the curve up to 2 h were reconstructed based on the experimental studies. For the first 15 min (15 m) after coronary occlusion, myocardial necrosis is not observed. At 40 min (40 m) after coronary occlusion, myocardial cell death develops rapidly and the myocardial necrosis is confluent.10 After this point, progression to necrosis is slowed considerably. The other parts of the curve showing myocardial salvage from 2 to >12 h from the symptom onset are reconstructed according to the data of scintigraphic studies.21,42,63 Efficacy of reperfusion is expressed as follows: ++++, very effective; ++, effective; +, moderately effective; ±, uncertainly effective; –, not effective.
with milder forms of myocardial ischaemia due to preservation of residual blood flow in the ischaemic area. Although not proven, this possibility sounds reasonable. Thus, it may be hypothesized that patients coming late after AMI onset may do so due to less dramatic symptoms which may be less coercive or perceived less threatening to seek urgent medical aid. Quite possibly, these patients may have preserved residual blood flow in the infarct-related artery which is responsible for milder myocardial ischaemia and less dramatic presentation. If so, then latecomers inherently may present a group of patients with AMI with highest chances of having extensive areas of viable myocardium in the area at risk that benefit mostly by mechanical reperfusion.

It is known that patients with AMI presenting late after symptom onset may have less or no chest pain that erroneously may be interpreted as a marker of no ongoing myocardial ischaemia. Attenuation of chest pain in patients with AMI may result from necrosis-related sympathetic denervation and does not necessarily reflect subduing of myocardial ischaemia. Previous studies have suggested that sympathetic afferent fibres mediate the sensation of cardiac pain in man.79 Experimental studies unequivocally have indicated that transmural myocardial infarction produces sympathetic denervation in the infarcted and non-infarcted myocardium apical to the region of necrosis demonstrated 90 min after coronary artery embolization.80 Although there is controversy whether myocardial ischaemia results in greater damage to myocardial sympathetic fibres than to working myocytes,81,82 it is tempting to suggest that sympathetic denervation reduces or abolishes pain sensation in the presence of evolving myocardial ischaemia. This has been demonstrated by the BRAVE-2 trial which showed considerable myocardial salvage in asymptomatic latecomers with AMI by PCI.83 From these facts, it is clear that the lack of chest pain may not exclude myocardial ischaemia and the viable myocardium, particularly in latecomers with AMI, and it should not be used as an index to rule out the PCI in these patients.

Concluding remarks

In this presentation, we summarized the experimental and clinical evidence confirming that in patients with AMI presenting late after onset of ischaemia, mechanical reperfusion results in considerable myocardial salvage. By emphasizing this fact, however, we do not mean to contest the concept of time dependence of myocardial necrosis following coronary occlusion or to dissuade the early coronary interventions in patients with AMI. On the basis of the existing experimental and clinical evidence, a portion of severely ischaemic myocardium succumbs to necrosis in a time-dependent and rapid way. At the early phase of rapid progression of ischaemic myocardium to necrosis, the earlier the application of reperfusion therapy, thrombolysis, or primary PCI, the greater the amount of myocardium salvaged. Experimental and clinical evidence indicates that a large amount of viable myocardium is still present in the area at risk in patients with AMI presenting late after symptom onset and considered ineligible for thrombolysis. This viable myocardium is salvageable given the primary PCI is used as a reperfusion therapy. For this reason, we strongly advocate primary PCI in patients with AMI presenting late after onset of myocardial ischaemia.

Conflict of interest: none declared.

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